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VASCULAR DISEASE

TWELVE MONTH PERSISTENCE OF POOR RESPONSE TO ASPIRIN IN PATIENTS WITH PERIPHERAL ARTERIAL DISEASE ENROLLED IN THE EFFECT OF LIPID MODIFICATION ON PERIPHERAL ARTERIAL DISEASE AFTER ENDOVASCULAR INTERVENTION TRIAL (ELIMIT)

ACC Poster Contributions
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Background: Poor response to aspirin (ASA) in patients with peripheral arterial disease (PAD) can be a major problem. Based on 1 measurement of arachidonic acid (AA) induced platelet aggregation, we previously reported that poor response to ASA (PNR) in PAD patients may be as high as 24%. Given possible non-compliance we tested the rate of persistent PNR. Additionally we evaluated the other clinically used measures to test aspirin response including the Platelet Function Analyzer (PFA)-100 epinephrine closure time and optical aggregometry with adenosine diphosphate (ADP).

Methods: PAD patients (n=74), on ASA who had platelet function testing (optical aggregometry with arachidonic acid (AA) and adenosine diphosphate (ADP), PFA-100 with collagen/ADP (cADP) and collagen/epinephrine (cEPI) cartridges) at baseline and follow up [6 months (n=70), 12 months (n=56), or both (n=54)] were included. PNR was defined as AA induced platelet aggregation >30%. Continuous variables are reported as mean ± SD.

Results: At baseline, 16.2% (n=12) had AA aggregation >30%. All 12 individuals were tested at 6 month and 8 of them at 12 month follow up. At 6 months, 7.1% (n=5) and at 12 months 5.3% (n=3) remained PNR. There were no differences in the PFA closure time between PNR and ASA responders at baseline (cADP 124±38 p=0.52, and cEPI 183.8±73 p=0.17), at 6 month (173±116.3 p=0.33, and 179.4±110.5 p=0.29), and at 12 month (168±114.4 p=0.56, and 165.7±116.6 p=0.5) respectively. No difference was found in ADP aggregometry between PNR and ASA responders (58.2±37.8 vs. 65.7±29.6, p=0.69) at baseline, 6 month (p=0.92), or 12 month (p=0.96). However, patients who were initially PNR and subsequently ASA responsive had significantly shorter cEPI times than patients who remained ASA responsive (125.5 ± 31.8 vs. 238.6 ± 81, p<0.001).

Conclusion: These data suggest that 1) 5.3-7.1% of PAD patients respond poorly to ASA over 12 month follow up with repeat measures; 2) AA-induced platelet aggregation is not consistent with the PFA closure time, even though both assays are widely used for measuring clinical response to aspirin and 2) ASA intake has a minimal impact on ADP induced platelet aggregation and PFA measurements in PAD patients